What is claimed is:

1. A method of treating an autoimmune or chronic inflammatory condition in a patient, said method comprising administering to the patient an antibody specific for CD30L that is capable of inhibiting the binding of CD30 to CD30L, wherein the antibody is administered according to a regimen of dose and frequency of administration that is adequate to induce a sustained improvement in at least one indicator that reflects the severity of the patient's condition, the improvement being considered sustained if the patient exhibits the improvement on at least two occasions separated by at least one day and further wherein the patient's condition is selected from the group consisting of psoriatic arthritis; arthritis nodosa; seronegative spondylarthropathies, and inflammatory bowel diseases.

2. The method according to claim 1, wherein the patient is a human.

3. The method according to claim 2, wherein the condition is ankylosing spondylitis.

4. The method according to claim 2, wherein the condition is psoriatic arthritis.

5. The method according to claim 2, wherein the condition is selected from the group consisting of Crohn's disease and ulcerative colitis.

6. The method according to claim 2, wherein the antibody is administered concurrently with a second agent that is an antagonist of TNFα, IL-1α, IL-1β or IL-4.

7. The method according to claim 6, wherein the second agent is an antagonist of TNFα selected from the group consisting of etanercept, p55 TNFR-Ig fusion protein and an antibody against TNFα.

8. The method according to claim 7, wherein the antagonist of TNFα is an antibody against TNFα, and further wherein said antibody is selected from the group consisting of infliximab, D2E7 and CDP571.

9. The method according to claim 2, wherein the antibody specific for CD30L is a monoclonal antibody.

10. The method according to claim 9, wherein the antibody specific for CD30L is a humanized antibody.

11. The method according to claim 9, wherein the antibody specific for CD30L is a human antibody.

12. The method according to claim 3, wherein the antibody specific for CD30L is a monoclonal antibody.

13. The method according to claim 12, wherein the antibody specific for CD30L is a humanized antibody.

14. The method according to claim 12, wherein the antibody specific for CD30L is a human antibody.

15. The method according to claim 4, wherein the antibody specific for CD30L is a monoclonal antibody.

16. The method according to claim 15, wherein the antibody specific for CD30L is a humanized antibody.

17. The method according to claim 15, wherein the antibody specific for CD30L is a human antibody.

18. The method according to claim 5, wherein the antibody specific for CD30L is a monoclonal antibody.

19. The method according to claim 18, wherein the antibody specific for CD30L is a humanized antibody.

20. The method according to claim 18, wherein the antibody specific for CD30L is a human antibody.

21. The method according to claim 6, wherein the antibody specific for CD30L is a monoclonal antibody.

22. The method according to claim 21, wherein the antibody specific for CD30L is a humanized antibody.

23. The method according to claim 21, wherein the antibody specific for CD30L is a human antibody.

24. An animal model for screening therapeutic agents, said animal model being characterized by:

(a) carrying genetic modifications that inactivate its p55 and p75 TNFα receptor proteins; and

(b) being genetically susceptible to experimentally-induced arthritis.

25. The animal model according to claim 24, wherein the arthritis to which the animal model is genetically susceptible is collagen-induced arthritis.

26. The animal model according to claim 25, wherein the animal model is a strain of mouse that is selected from the group consisting of DBA/1, BUB and B10.Q or a strain of rat that is selected from the group consisting of DA, BB-DR and LEW.

27. The animal model according to claim 26, wherein the strain of mouse is DBA/1, and further wherein the DBA/1 strain of mouse has double-null mutations in its p55 and p75 TNFα receptor genes.

28. A method for screening a candidate therapeutic agent to determine its efficacy in treating an autoimmune or chronic inflammatory condition that is resistant to treatment with a TNFα inhibitor, said method comprising inducing arthritis in the animal model of claim 27, administering the candidate therapeutic agent to said animal, and

determining that the agent is efficacious if the severity of said animal's arthritis is reduced after the candidate agent has been administered.